

**CODING DISABILITY:
A PURE DYSGRAPHIA**

CENTRE FOR NEWFOUNDLAND STUDIES

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Coding Disability: A Pure Dysgraphia

Submitted by



Joan Alfreda Lawrence

B.Sc., Memorial University of Newfoundland,
in partial fulfillment of the requirements
for the degree of Master of Science.

Department of Psychology

Memorial University of Newfoundland

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St. John's

Newfoundland

Abstract

In the present investigation, an attempt was made to identify a learning disability defined by a characteristic pattern of inconsistencies between written and oral school performance. Children with at least average verbal skills were described as being unable to produce coherent, detailed written passages, and their WISC-R Coding scores were depressed significantly in comparison to their other subtest scores. Labelled as Coding Disability (CD), the disorder was postulated to be a form of apraxic dysgraphia occurring in the absence of verbal/linguistic deficits. Twelve children whose Coding scores were at least 3 points lower than their Vocabulary scores were selected for the Coding Disabled group. They were matched on age and sex with children who showed minimal subtest scatter. The Similarities, Vocabulary, Block Design, and Coding subtests, the Peabody Picture Vocabulary Test, and a modified version of the Gates-MacGinitie reading comprehension test were administered to both groups. Because the Coding Disability was postulated to be neurologically mediated and independent of external influences, measurements were repeated under low and high motivation. It was predicted that, under high motivation, the CD children would improve significantly on all scores except Coding and Written recall. The Control children were expected to improve on all scores. This hypothesis was confirmed; change scores differentiated the two groups only on the Coding and Written recall tests. The findings point to

the existence of a group of children whose poor writing skills are
hardwired and impervious to motivational influences.

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Unexplained underachievement in school children has long been recognized as a major concern to educators and other professionals in the field of child behaviour. Over the past 15 to 20 years, increased research in the area has led to greater understanding of many problems once identified as laziness, poor attitude, and stupidity. While there remain children who are unmotivated, uninterested, or dull, others, who may once have fallen into one of these categories, have been found to be suffering from hardwired, neurologically mediated defects now known as learning disabilities. Initially a 'catch all' diagnostic label subsuming any type of learning disorder, learning disabilities have come to be differentiated by and categorized under three general headings - the dyslexias or specific reading disorders, the dyscalculias or mathematics disabilities, and the dysgraphias or disorders of writing. Authorities in the area link these specific learning disabilities to specific deficits by conceptualizing them in terms of a matrix which relates academic achievement skills to neurocognitive functions. It is postulated that reading/spelling disorders are associated strongly with deficits in language and sequential/analytic skills, and are also influenced by poor visuo-spatial abilities. Arithmetic problems are thought to result from impaired visuo-spatial and sequential/analytic skills. Dysgraphias, on the other hand, are primarily associated with poor motor planning and execution skills, plus deficiencies in sequencing and visuo-spatial abilities.

By far the most common and most widely studied of the learning

disabilities are the dyslexias. These can be subdivided into two main groups, reading disorders and spelling disabilities (Boder, 1973).

The former seem to involve a central language processing dysfunction most evident in oral reading. It manifests itself in a variety of ways, including poor word attack skills, improper pronunciation, interchanging of words with similar meanings, and guessing at words on the basis of a few letters. The spelling disabilities appear as letter sequencing mistakes, letter reversals and rotations, and spelling errors characterized by phonic correctness. These symptoms suggest that the spelling disorder is due to a visuo-spatial and sequential/analytic skills deficit.

Children with specific mathematical deficits rarely show any difficulties with reading or spelling, although some individuals do display both math and reading type disabilities. Relatively little research has been done into the dyscalculias, but Rourke and Finlayson (1978), Rourke and Strang (1978), and Rourke (1978a, 1978b) have discovered that math disabilities may result from either visuo-spatial deficits or poor language development. After noting problems with ordering and sequencing of numbers, in addition to difficulty in reading and understanding written arithmetic, Kinsbourne (1977) concluded that some form of symbol decoding dysfunction was present.

As is the case with the math disabilities, little research has been done into the disorders of writing. It has been found that dysgraphias rarely occur alone, but are most commonly found in

combination with other forms of learning disabilities, particularly dyslexias or dysphasias. Naidoo (1972) has isolated two such subgroups of dysgraphia. Although they manifest themselves in a similar manner, the subgroups may be differentiated on the basis of their apparent underlying defects. The first type of writing difficulty appears to be caused by an inherent malfunction in linguistic processing abilities, specifically, phonetic analytic skills (Naidoo, 1972; Hecaen, and Albert, 1978). This disorder usually involves bizarre, dysphonetic spelling errors characterized by omissions of entire syllables or inclusions of unnecessary letters and phonemes. Because the spelled word is so unlike the original, children suffering from this disorder are often investigated for hearing problems. They also experience extreme difficulty with oral reading, often excluding parts of words or including sounds that should not be there. Reading comprehension and copying of words from printed text is usually intact.

Another form of dysgraphia, also characterized by spelling mistakes, is that which appears to stem from defects in visuo-spatial skills. Children affected by this problem appear to be unable to perceive the proper orientation and formation of printed symbols. Consequently, they produce or reproduce letters and numbers in a distorted manner. Such distortions include severely slanted writing, perseveration (especially in graphemes with many loops), and words broken up by improperly placed blanks. Spelling mistakes are more likely to be phonetically correct, since there are no apparent

linguistic phonemic deficits. Not surprisingly, this form of dysgraphia is most commonly accompanied by disorders of arithmetic, in which the child cannot seem to organize or produce figures on the page in a logical manner. Consequently, although the child may understand the concept s/he is trying to demonstrate, his/her haphazard manner of writing down the numerals often results in his/her performing the operations on the wrong numbers. Such arithmetic disability may be closely related to disorders of writing, visual motor integration, and short term memory. Siegel and Feldman (1983) found that some children with learning disabilities were deficient on written and oral short term memory tests, suggesting that their math difficulties stemmed from an inability to remember the numbers or operations at hand. Others were deficient in visuo-motor co-ordination which may have resulted in problems with writing mathematical calculations and solutions. This writing deficit may account for the haphazardly written math schoolwork observed by Naidoo (1972).

There remains a group of children who do not fit the description of the well known forms of learning disabilities and who are often considered to be simply 'lazy'. Recent evidence (Heilman and Valenstein, 1979) suggests that some lazy children are not really unmotivated, but may suffer from a hitherto unrecognized form of learning disability manifesting itself as an inability to plan, organize, and execute voluntary graphomotor movement. Although its existence has not yet been documented, Heilman and Valenstein have postulated that the disorder is related to apraxia and may be

classified under the broad heading of dysgraphia. If it occurs in the absence of any other form of learning or academic disability, it may be the only 'pure' dysgraphia.

While no empirical evidence yet exists to provide a comprehensive behavioural description of this apraxic dysgraphia, the following clinical picture emerges from past studies of children with written output difficulties (Heilman and Valenstein, 1979; Barkley, 1981; Levine et al., 1981). The affected children appear to be bright in class and to have no problems expressing themselves verbally. Consequently, they are thought to be of at least average intelligence and to have no conceptual or comprehension problems. They are, however, markedly dysfluent in their written expression. The children do well on verbal performance assessment and multiple choice/fill in the blank type exams which require minimal writing, but in any situation requiring written output, quality of work falls drastically. They display very clumsy, slow, and ill formed writing. They may be unable to finish exams within the given time limits, and what they do write may be unreadable. Some children, determined to finish their schoolwork within the allotted time, may resort to giving very brief answers characterized by poor ideational content and word omission. Confusion of meaning results. Others, finding writing an unpleasant and laborious chore, refrain from writing at all. They write one or two sentences in response to essay exam questions and rarely attempt written homework. The contrast between their obvious understanding and intact knowledge of the material, and their apparent refusal to

put their knowledge onto paper, results in teachers labelling them as 'lazy' or unmotivated (Barkley, 1981).

The present investigation will address itself to isolating this hypothesised group of dysgraphic children. Because the postulated problem appears to be specific to graphomotor movement, it is reasonable to predict that dysgraphic children would display adequate performance on all of the WISC-R tests except Coding which involves a form of writing (Barkley, 1981). Accordingly, this study will select a group of such children, to be called Coding Disabled (CD), on the basis of their low Coding scores in contrast to their other WISC-R scores. Their performance on various written and verbal tasks will be compared with that of children who show minimal subtest scatter. It is predicted that the CD children will perform as well as the others on all tasks except those requiring written output, on which they will be significantly worse. If, like all learning disabilities, Coding Disability, or apraxic dysgraphia, can be thought of as being caused by a neurocognitive dysfunction, it should be minimally affected by external variables such as motivation. Therefore, in one testing session under conditions of increased motivation, the CD children would be expected to improve their scores on all except the written tasks, while the Control children would improve on all scores. Presumably, with intensive practice and high motivation, even the CD group's written performance would show some improvement.

Empirical Evidence for Apraxic Dysgraphia

Coding Disability, as it has been defined for the purposes of this study, has not yet been identified as a separate learning Disability and has not been the subject of specific study. In reviewing the literature, it becomes evident that many researchers have encountered this disability, or similar syndromes, in their studies of general writing disorders. As a result, in attempting to provide background into the nature and etiology of the proposed Coding Disability, it is necessary to review related work in the areas of dysgraphia, agraphia, and apraxia.

Levine et al. (1981) examined what they refer to as "developmental output failure" in terms of the various types of deficits or problems directly influencing a child's lack of written production. Not unlike Naidoo, these researchers demonstrated the existence of three distinct areas of writing difficulty. A small number of their dysgraphic subjects exhibited expressive language problems roughly analagous to those found in the phonetic dysgraphia identified by Naidoo's research. The children were markedly dysfluent, articulated poorly, and were hesitant to name objects verbally or to read simple sentences. Others displayed visual retrieval and letter sequencing problems similar to those experienced by Naidoo's visuo-spatial dysgraphics. These children had difficulty

in storing and retrieving symbol sequences, and they were unable to remember and to reproduce alphanumeric symbols and geometric designs accurately.

By far the most common problems, the existence of which lends support to the apraxic agraphia hypothesis of Heilman and Valenstein, concerned fine motor co-ordination. From 50 to 72 percent of the children tested by Levine et al. had some form of apraxic deficit affecting their ability to write efficiently. Fine motor skills such as bead stringing, sequential finger opposition, and rapid line drawing were clumsy, unco-ordinated and spastic. Pencil control was poor, with the most common inappropriate writing techniques including awkward pencil grip, extreme pencil pressure, and fist contortion. Combined with the fine motor deficits, this plethora of pencil control difficulties led to illegible, slow, and tiresome writing. In view of these assorted motor problems, it is not surprising that most of the children had inefficient or impaired graphomotor patterns and rhythms.

An accompanying motor difficulty found to affect writing was finger agnosia. The children with poor proprioceptive kinaesthetic feedback were unable to write efficiently unless they constantly visually monitored their hand movements. Even then, their writing was extremely slow. Additionally, all children with finger agnosia also had fine motor co-ordination problems, but whether or not there existed a causal relationship between the two disorders was unclear.

In summary, the educational literature suggests that dysgraphia can be divided into three distinct and separate disorders of writing. The phonetic/linguistic and visuo-spatial dysgraphias are reasonably easy to understand because of their marked and obvious patterns of concrete errors. Consequently, they tend to be more often diagnosed, accepted, and treated as learning disabilities than are the apraxic dysgraphias which display no such tangible symptoms. Because this latter type of dysgraphia manifests itself as a lack of concrete output, it is less likely to be noticed and diagnosed as a learning disability.

Further support for the existence of a motor dysgraphia is offered by the neuropsychological studies of agraphia and apraxia (Hecaen and Albert, 1978; Marcie and Hecaen, 1979; Heilman et al., 1973, 1974). Although this research does not deal directly with school related learning disabilities, it does demonstrate the existence of an apraxically mediated form of writing disorder.

While writing may be considered an alternate form of spoken language, in the final analysis, it is a praxic as well as a linguistic activity. It is obvious that any disorder affecting praxis would inevitably have a detrimental effect on written production. Moreover, a limb kinetic type of apraxia not affecting spoken language, but influencing hand movement, would produce exactly the effect postulated by Heilman and Valenstein (1979). Good verbal skills would be intact, but the individual would have severe

difficulty organizing the movements necessary to put his thoughts on paper.

Hecaen and Albert (1978) and Marcie and Hecaen (1979) have argued that apraxia interferes with the motor ability to form symbols. Such interference results in illegible letter formation and/or inversions or substitutions of the desired graphemes. They claim that the major clinical syndromes associated with this problem would be agraphia with ideomotor apraxia and apraxic agraphia with alexia. The former involves bimanual agraphia with apraxia limited to the non-dominant hand. This syndrome is of little interest here, save that it demonstrates the simultaneous occurrence of apraxia and agraphia. It is not clear why the dominant hand is not agraphic, or if there exists a causal relationship between the two disorders within the non-dominant limb. Of greater relevance is the case of apraxic agraphia with alexia (ie. a combination of motor related writing disorder with a reading disability). In this syndrome, the agraphia is characterized by apraxically mediated distortions and inversions of graphemes, as well as by paragraphic and spelling errors resulting from the alexia. The authors argue that the agraphia is the result of defects at both the linguistic and gestural organizational levels.

Heilman et al. (1973) present an interesting case of a left handed man who habitually wrote with his right hand and who, as the result of brain damage, developed an ideomotor apraxis in that hand. All linguistic functions were normal, but he was unable to carry out

voluntary motor actions, including writing, correctly with his right hand. Only with great effort was the patient able to write his name, and his attempts to write anything else revealed a consistent pattern of omissions of word parts and grapheme substitution. As a rule, he was able to complete the first 2 or 3 letters of any word correctly, but was unable to continue. However, his oral spelling was flawless. A similar combination of apraxia and agraphia in a right hander was reported by Heilman et al. (1974), leading the authors to conclude that there was a causal relationship between apraxia and agraphia.

Demonstrating the co-existence of apraxia and agraphia covers only half of the problem at hand. While the hypothetical Coding Disabled children are thought to exhibit a combination of these disorders, it must be remembered that their writing disorder is not accompanied by any apparent linguistic malfunction. As has been stated previously, dysgraphias rarely occur without some form of verbal language problem (Heilman and Valenstein, 1979; Barkley, 1981; Marcie, 1977), although Siegel and Feldman (1983) have found writing disorders in combination with arithmetic and short term memory problems. It remains, therefore, to demonstrate the existence of an agraphia/dysgraphia which is independent of any form of verbal linguistic deficiency.

One of the earliest known examples of an agraphia without aphasia was reported by Gordinier (1899). In one patient, oral reading and spelling, as well as silent reading and the comprehension thereof,

were intact. Recognition of numerals and letters was quick and accurate, as was mental computation and sight reading of contextually isolated words. The patient's only difficulty was her total inability to write. Although she held the pen correctly and attempted the writing movement, the end product consisted of a series of repeating loops. She was unable to write or print a single letter, either spontaneously, or by direct copying from printed text. Gordinier concluded that aphasia and this form of apraxic agraphia must be physiologically distinct syndromes, thus implying that one can exist without the other.

More recently, on the basis of the study of six patients displaying 'pure' agraphia, Dubois et al. (1969) suggested that the disorder was not a unitary entity, but could be broken down into two dysfunctions of basic writing abilities. The subjects of the study exhibited a significant number of written spelling errors while maintaining flawless oral spelling skills. Deeper analysis of these errors revealed that they were more common in the middle of words than at the ends, and that they were restricted to mistakes in grapheme selection and combination. Letter formation and the linguistic/phonetic aspects of the words, such as letter to sound correlation, remained intact. Finally, for half of the patients, writing in manuscript was significantly worse than arranging anagram letters (letters stamped onto blocks or tiles). Dubois et al. argued that pure dysgraphia results from the combination of a defect in grapheme selection ability with a graphomotor specific apraxia which

is unrelated to constructional apraxia, and which results in distortion and disorganization of writing in space and time.

Similar results were presented in a case study reported by Assal et al. (1970), who treated a patient suffering from a pure agraphia stemming from atherosclerosis of several of the major arteries of the brain. Upon examination, it was revealed that all oral language functions, including spelling and reading, were intact. Mental arithmetic computation and visual recognition of designs, faces and body parts was also unaffected. There was no apparent praxis of any type. Aside from the writing problem, only intellectual functioning, as measured by the WAIS, showed any evidence of deterioration from the estimated premorbid level. Writing, especially narrative writing, was grossly affected. The patient's productions displayed poverty of ideational content and general incoherence. In both spontaneous and dictated writing, bizarre spelling mistakes and total disruption of punctuation skills were found. Direct copying from printed text was also inaccurate. However, the problem was not limited only to expression involving graphomotor movement; the patient also had difficulty constructing words with letter blocks. Consequently, Assal and associates (1970, 1978) concluded that the agraphia was a function of a basic defect in encoding ability, as opposed to some type of praxic disorder.

On the basis of their studies of agraphia to dictation, Weigl and Fradis (1977) postulated that agraphia results from a disturbance of

transcoding from verbal words into graphemes into graphomotor movements. They advanced a three stage hypothesis which provides an appealing logical basis for explaining agraphia with or without aphasia. It can be argued that when agraphia and aphasia occur in conjunction, the transcoding function breaks down at its earliest point - understanding the words. If an individual cannot understand the words he hears, he has difficulty in using them to write coherently. When agraphia occurs in the absence of a speech disorder, the coding process breaks down at one of two points. It may, as Assal et al. have speculated, fail at the second stage where words are being transcribed into graphemes. This accounts for agraphia occurring in the absence of disorders of speech, reading, or movement. Alternatively, the problem may be a purely praxic one in which the graphemes are not efficiently recoded into graphomotor gestures. This defect should result in a pattern of symptoms very similar to those displayed by the hypothetical group of Coding Disabled children.

Despite the dearth of literature on the subject, the few relevant studies offered suggest that writing disabilities can and do exist in the absence of reading or speech problems. Such deficits are probably mediated at the neurological level by some form of praxis or encoding malfunction.

Neurological Correlates of Pure Agraphia

Much of the scientific study of writing dysfunctions has been done from the neurocognitive viewpoint. While the dichotomy between the visuo-spatial and phonetic/linguistic subtypes persists into this literature, there is conflict among the experts about which, if either, is 'true' agraphia. It is the opinion of one group that the visuo-spatial form is the only real agraphia and that it results from right lateral lesions which disrupt the spatial organizational processes prerequisite to writing. Supporters of the phonetic/linguistic subtype feel that writing is an outgrowth of spoken language and that pure dysgraphia can only result from a disorder of language processing caused by left hemisphere brain damage. A third much smaller faction argues that true agraphia is the result of neither linguistic nor visuo-spatial processing dysfunctions. From this reductionistic point of view, the only true agraphia is the apraxic form which is, literally, a specific disability of graphomotor movement. The other forms of agraphia are considered not to be agraphia at all, but simply symptoms of dyslexia, dysphasia, and visuo-spatial malfunction. In view of the postulated major role of the apraxic syndrome in the type of agraphia under investigation, the specific causes of related praxic disorders must be considered.

Given Heilman and Valenstein's (1979) description of apraxic agraphia, it seems that they were referring to the limb kinetic form of movement disorder. First identified by Leipman (1900), limb kinetic apraxia is characterized by impaired position sense, spasticity, and difficulty in performing purposeful voluntary movements. Such a disorder would have pronounced effects on fine motor tasks such as writing. There has been very little study into the neurological basis of limb kinetic apraxia, and what has been done has failed to produce any clear evidence for exact localization of function. Leipman speculated that the problem lay in damage to the lateral areas of the dominant hemisphere. Brown (1972) cites Denny-Brown (1938) and Neilson and Friedman (1941) as placing the lesions in the frontal lobes and the cortical motor area. Luria (1966) suggested that it might be attributed to premotor lesions in the post central regions of the cortex.

Another form of apraxia which may be related to agraphia is the ideomotor variety, which is characterized by an inability to carry out simple movements upon command. Though the patient understands what is required, s/he appears to be able to transcode only a part, if any, of the ideation into motor movement. This results in bizarre, but vaguely relevant, approximations to the desired gesture. Within the framework of writing disorders, this form of apraxia would be exemplified by the patient who could write only parts of words correctly. Leipman argued that ideomotor apraxia is caused primarily by lesions in the area of the dominant supramarginal gyrus.

Additional lesions of the corpus callosum would result in similar syndromes related to the non-dominant hand, the legs and the facial area. This localization was uniformly accepted by Leipman's contemporaries and remains in effect to the present day (Brown, 1972).

According to Heilman (1979), the general consensus of opinion is that apraxic agraphia is due to a combination of callosal dysfunction and dominant hemiparesis. Geschwind and Kaplan (1967) and Gazzaniga et al. (1967), in their studies of commissurotomy patients, report a direct relationship between callosal lesions and apraxic agraphia in the non-dominant hand. It appears that, prior to lesioning, the dominant hemisphere uses the corpus callosum as a means of communicating with the non-dominant hemisphere, and assisting it in the mechanics of writing. When the callosum is severed, the non-dominant hemisphere no longer receives instructions from the dominant half, thus rendering it incapable of activating the non-preferred hand to write. Writing remains intact in the dominant hand because the neural connections between it and the hemisphere controlling writing remain intact. These studies imply that the dominant hemisphere functionally controls writing in either hand, using the corpus callosum as a means of conveying instructions to the non-dominant when it is necessary to write with the non-preferred hand. Consequently, it can be argued that lesions to the dominant hemisphere or severe lesions to the callosum can result in agraphia.

Notwithstanding that these studies confirm the important role of

the dominant hemisphere and the corpus callosum in mediating written output, they are not particularly applicable to most victims of agraphia whose corpus callosa are, as a rule, intact. Reilman et al. (1973, 1974) report cases of right and left dominant individuals who developed apraxia and agraphia subsequent to the sudden onset of hemiparesis of the dominant lobe. Neurophysiological assessment revealed lesions to the dominant half of the brain. It was argued that damage to the lobe which controls handedness destroyed the engrams for writing and, in doing so, resulted in agraphia. The role of the corpus callosum was demonstrated by the case of the right hemisphere dominant man who habitually wrote with his right hand and who became agraphic in that hand. According to prevalent dominance hypotheses, it would be expected that the damage to the man's right hemisphere would result in left, not right hand, agraphia. This case was thought to parallel the commissurotomy studies insofar as the corpus callosum was used to transmit writing instructions from the right to the left side of the brain. When the right half was damaged, messages no longer crossed into the left side, so right hand agraphia occurred. There is a significant degree of similarity between this localization of agraphia and Leipman's conclusions about the origins of Ideo-motor apraxia.

Gordinier (1899) specified the exact location of function for writing in each hemisphere. He stated that the writing center for the right hand (left hemisphere dominant) is at the base of the second left frontal convolution of the brain and in the corresponding area of

the right hemisphere for the left handed. He also asserted that, should this particular area be destroyed or damaged, the inevitable result would be "pure motor agraphia without aphasia and with no paralysis of the arm...(Gordinier, 1899)". He cited, in support of his theory, the results of an autopsy performed on a right handed woman who was totally incapable of writing. The autopsy report stated that, in the second left frontal convolution, there was found a small tumor occupying exclusively the white matter of that part of the brain. No other brain region or cognitive/linguistic function was affected or involved. As a result of the specificity of the tumor site, Gordinier felt confident that there existed a separate, distinct, and exclusive motor center for writing.

Like Gordinier's agraphia, pure agraphia is thought to result from lesions of the dominant frontal lobe (Benson, 1979; Marcie and Hecaen, 1979). Although it was impossible to specify the exact site of the postulated lesions, Dubois et al. (1969) reported that 4 of 6 of their pure agraphia patients appeared to suffer from damage to the dominant frontal lobe. Assal and associates' (1970) patient suffered from a vascular condition (atherosclerosis and stenosis) which the researchers speculated to be affecting a major artery in the left frontal lobe, resulting in lesion and agraphia. Again, it was impossible to be definite about the existence of this lesion or to specify exactly where it was situated. Laine and Marttila (1981), however, reported a similar case of pure agraphia in which medical tests specifically implicated a vascular lesion in the area of the

left internal capsule and caudate nucleus.

In contrast to this body of literature suggesting the left frontal lobe's involvement in pure agraphia, Sugushita et al. (1980) argued for the primary role of the corpus callosum in the etiology of the disorder. Their patient underwent surgery which necessitated a two thirds severing of the truncus; he subsequently developed agraphia without apraxia. The authors claimed that this result suggested that the callosum does play some major part in mediating pure agraphia.

When these neurological studies are considered as a group, a consistent pattern emerges. The evidence suggests that agraphia/dysgraphia does exist in the absence of other disorders of speech and reading. Furthermore, such writing disabilities may occur in two forms, the pure dysgraphia which is totally independent of any other disability, and the apraxic agraphia which is associated with disturbances of graphomotor skills. Both forms, as well as the various related types of apraxia, appear to be mediated at the neurological level by the frontal lobe of the dominant hemisphere.

The current study will attempt to demonstrate the existence of a pure dysgraphia, Coding Disability, in school children.

Method

Subjects

Subjects were obtained through the co-operation of the Diagnostic and Remedial Unit, MUN. This is an agency to which children are referred for identification and treatment of a variety of academic and school related problems ranging from behavioural, emotional and social disorders to mental retardation and learning disabilities. An exhaustive search of all past and current files of the unit was conducted, and all children whose records contained a previously administered WISC-R test were considered for inclusion in the study. Because the participants were to be retested with parts of the WISC-R, children whose present age exceeded the norms covered by the WISC-R were immediately excluded. The remainder were then screened into two groups on the basis of the discrepancy between the Similarities, Vocabulary, Block Design, and Coding subtests of the prior Wechsler tests. Children whose Coding scaled score met a criterion of being at least 3 points lower than their scores on the other three subtests were placed in the Experimental or Coding Disabled (CD) group. Children whose low Coding score was judged by the previous examiner to be the result of some extraneous factor such as physical handicap, tension or other emotional state were excluded from the study.

Comparison (C) subjects were then chosen from among a pool of children whose scores on all four subtests fell within 2 points of each other; they were matched with the CD group on the basis of age and sex. At the end of the selection process, each group consisted of 12 boys with mean ages of 12 years 1 month and 11 years 11 months for the CD and C groups, respectively.

Materials

The WISC-R Similarities, Vocabulary, Block Design, and Coding subtests were used as general measures of intellectual and graphomotor skills. Because of the heavy reliance of the Wechsler Vocabulary subtest on good verbal expressive skills, the Peabody Picture Vocabulary Test (PPVT), Form L, was used as an auxiliary non-verbal measure of vocabulary. The Gates-MacGinitie Reading Tests, Levels C, D, E, and F, Form 1, were used in order to assess verbal and written academic output. For each paragraph in the comprehension section, a checklist of the main ideas was constructed for rapid evaluation during verbal recall.

Design

Two groups of subjects were tested with 8 repeated measures (Test) under 2 levels of motivation. Two orders of test presentation (WO and OW) were used for the Oral and Written recall of paragraphs. Order was counterbalanced and matched between groups.

Procedure

Testing was completed in two sessions each of 75 minutes duration. Subjects were tested individually and each child experienced all experimental manipulations.

Upon first meeting, each subject was told that he was being tested in order to determine how well he could do certain types of school work. Once a rapport was established, the Similarities, Vocabulary, Block Design, and Coding subtests of the WISC-R, and the PPVT were administered and scored in the standard manner. Having completed these tests, the child was allowed a 5 minute break during which he was free to get a drink or a snack, go to the washroom, play with toys, or talk with the experimenter. The age appropriate form of the Gates-MacGinitie Comprehension test was then administered in a slightly modified manner. Assessment of each paragraph was done

separately. The child read the paragraph and answered the accompanying multiple choice questions in the standard manner, after which the paragraph was concealed and the child was required to produce both written and verbal recall of the selection. Verbal recall was cued with the phrase "Tell me everything you can remember about that story." and was simultaneously evaluated using a checklist of main ideas. One point was scored for each chunk of information remembered (see Appendix A). Written recall was requested with "Write down everything you can remember about that story." and was graded using the same checklist of main concepts. Once both verbal and written recall had been completed, the child went on to do the next paragraph in the same manner. Order of recall was counterbalanced with half of the children doing verbal, then written recall and the other half doing the written recall first. Pilot data revealed that the optimal length of time for this testing period was 40 minutes. After that point, the children tended to become bored and/or tired. Consequently, the testing session was terminated 40 minutes after the introduction of the comprehension test. At the conclusion of the session, the child was informed that he would be returning the following week to complete more tests similar to those he had just done.

At the outset of the second testing session, the child was given his scores from the previous week and was told that if he could improve his scores on those same tests, he would be allowed to play with a tabletop video game. In order to maximize motivation, reward

increased with degree of improvement of test scores. The child was informed that an improvement of 10% would earn the opportunity to play 1 game, while a 20% improvement earned 2 games, 30%, 3 games, and so on. This system was explained to the child and he was told exactly how many points were required for each game for each test. All tests were then administered in the same manner as the previous week. Reward was administered immediately after the completion of the test session and any questions which the child had about the study were answered.

Results

Separate stepwise linear discriminant analyses were performed on the groups' mean test scores under normal and under high motivation, and on their mean change scores. All three analyses revealed, in accordance with the hypothesis, that only the Coding and/or Written recall scores significantly differentiated between the two groups.

The analysis revealed that only the Coding test significantly differentiated the CD from the C group under low motivation ($F=12.62$, $p<0.002$) and under high motivation ($F=29.55$, $p<0.0001$). Group differences failed to reach significance on all other tests. Figure 1 illustrates mean test scores for each group under low motivation, while Figure 2 displays these mean scores under the high motivation condition.

Analysis of the mean change scores revealed that Groups were significantly different on the Coding test ($F=13.34$, $p<0.001$) and on Written recall ($F=6.97$, $p<0.01$). The generated discriminant function allotted a discriminant coefficient of 0.79504 to the Coding test and one of 0.53646 to Written recall. This function proved to have 75% accuracy in predicting group membership. Figure 3 illustrates the differences in mean change scores.

Chi squared analysis of order of presentation and performance on Oral and Written recall failed to show a significant order effect ($p>0.30$).

In Figures 1 to 3, abbreviations are used as follows: Sim - Similarities, BLD - Block Design, Voc - Vocabulary, Cod - Coding, PPVT - Peabody Picture Vocabulary Test, WRc - Written Recall, ORc - Oral Recall, MC - Multiple Choice.

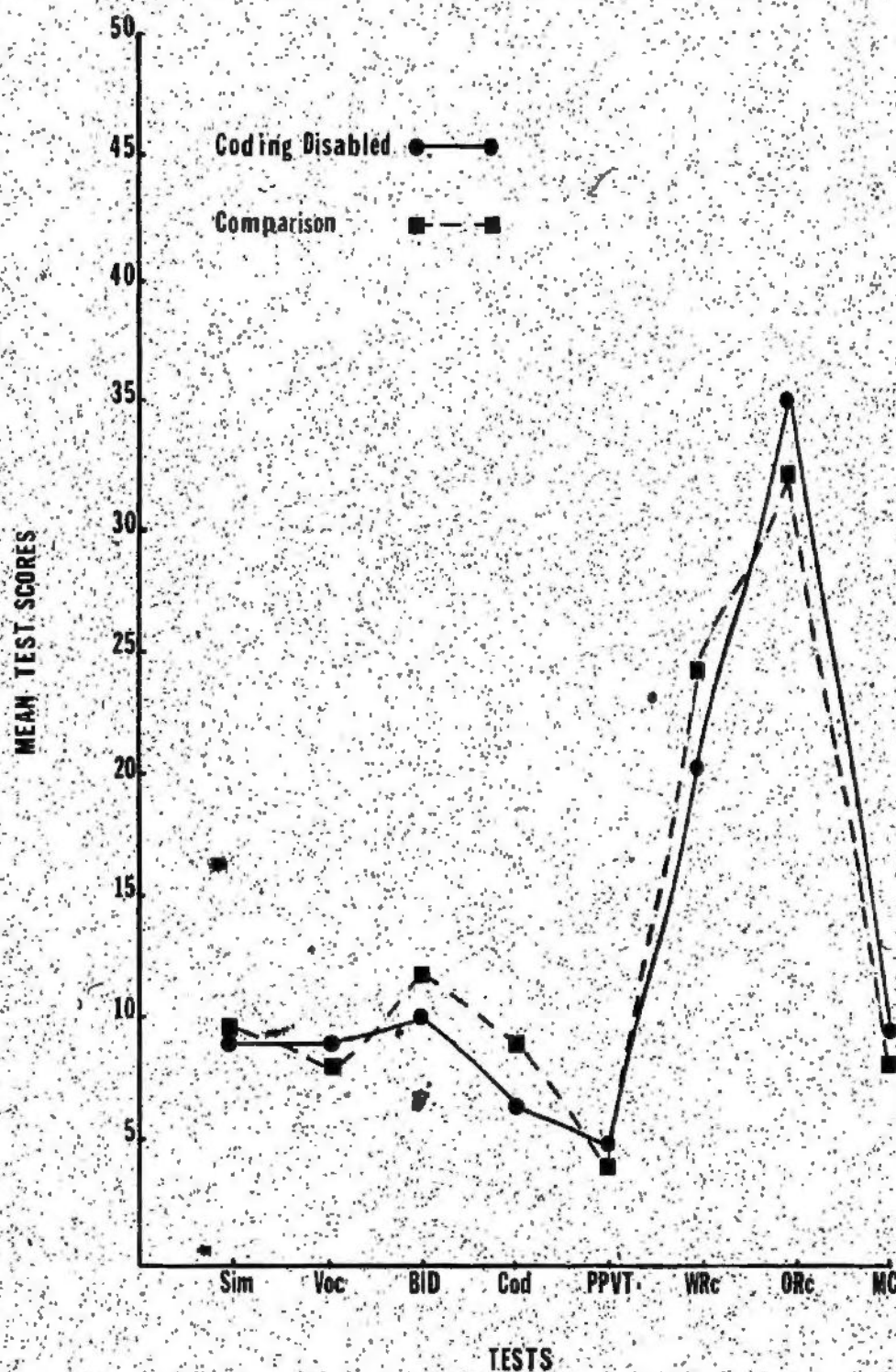


Figure 1. Mean group test scores under normal motivation.

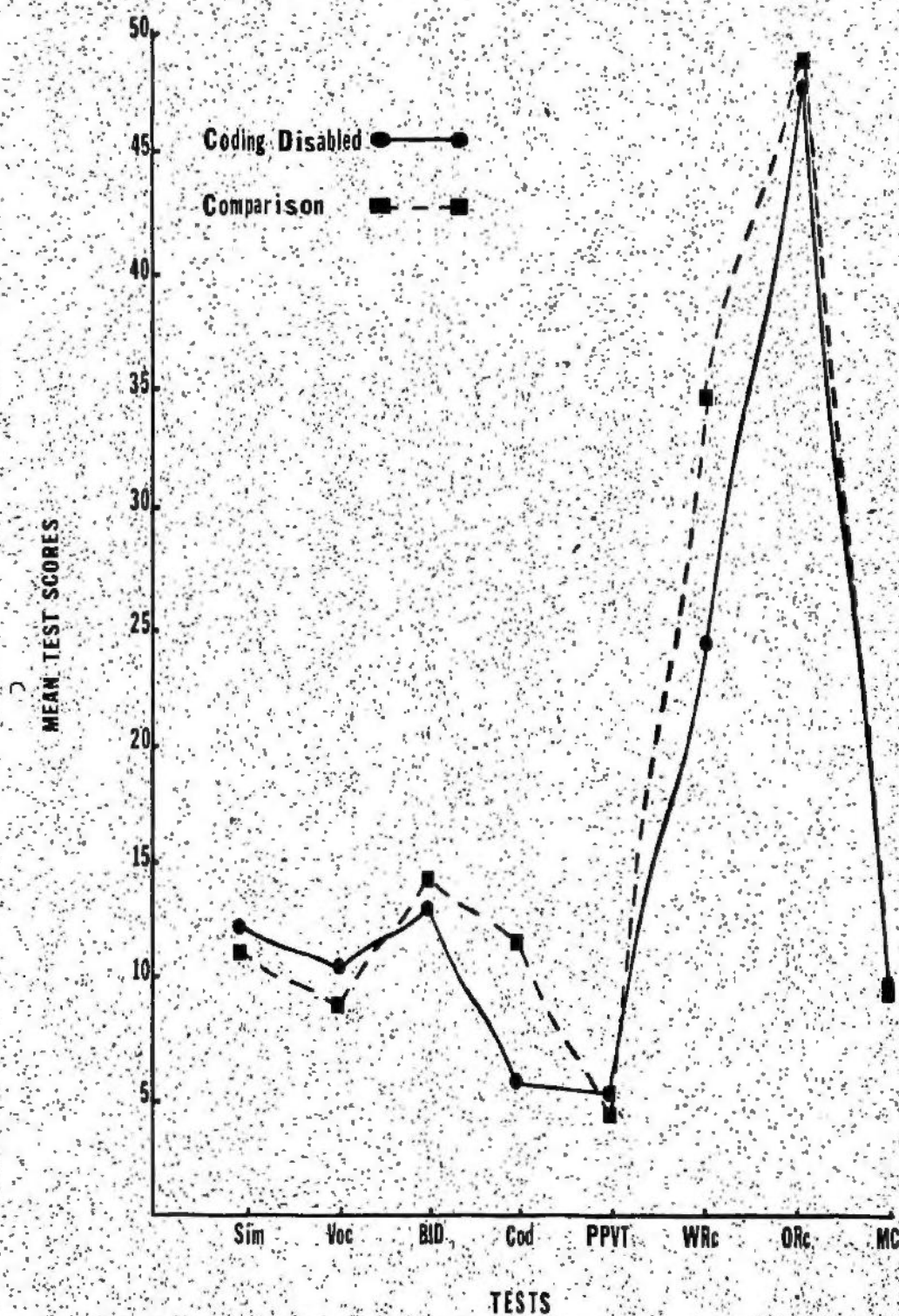


Figure 2. Mean group test scores under high motivation.

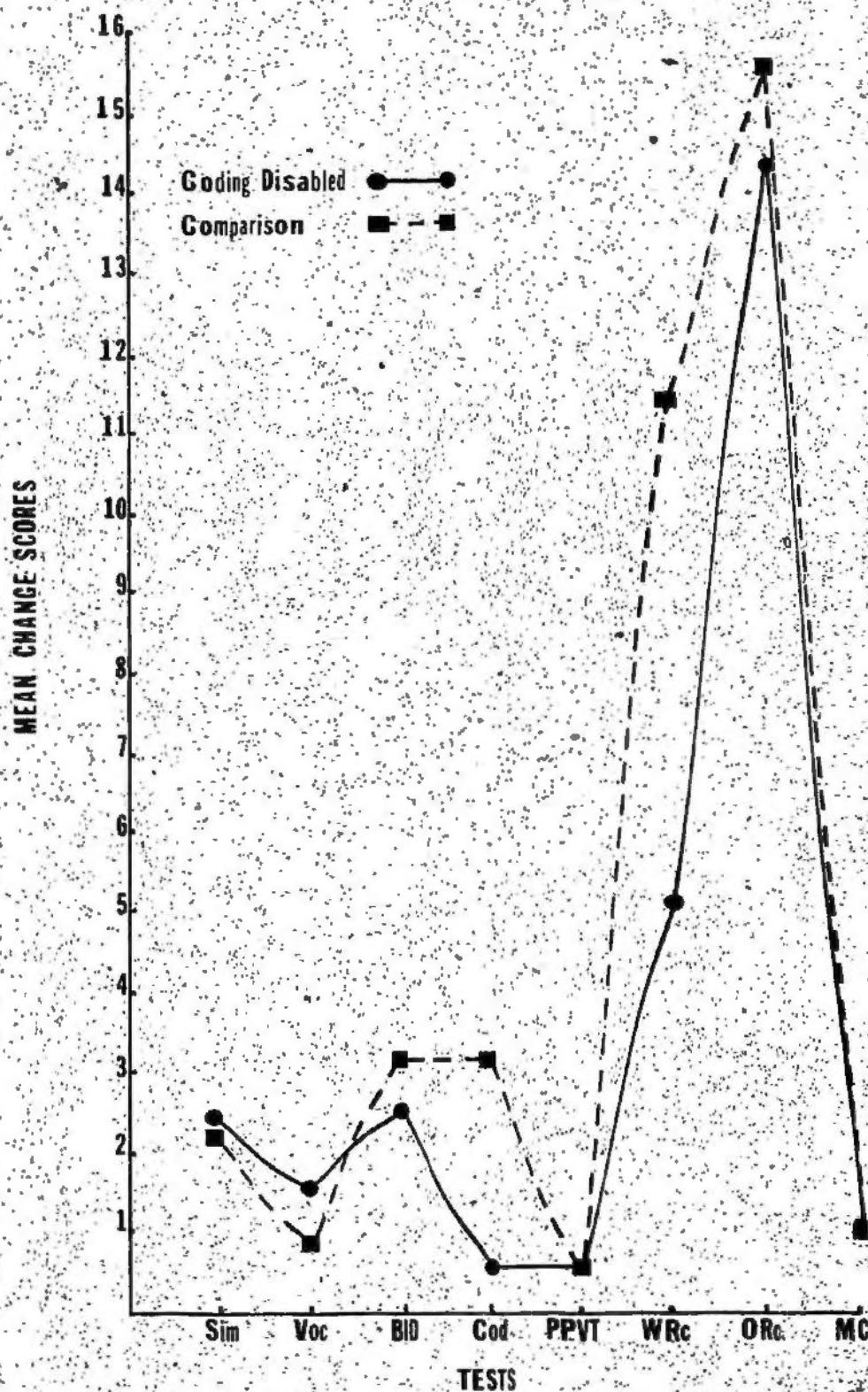


Figure 3. Mean group change scores.

Discussion

The results of the study confirm the hypothesis that WISC-R Coding scores which are significantly depressed in comparison to other subtest scores are associated with poor written output. Under both normal and high motivation, children who showed large discrepancies between their Coding and Vocabulary scores tended to perform as well as other children on oral and object manipulation tasks, but less well on those involving written output. Increasing motivation had little effect on the written performance of the CD children, although it facilitated performance on other tasks. As expected, the Comparison children improved their scores on all tasks under increased motivation. It may be concluded that the writing deficiencies displayed by the CD group did not originate from a motivational problem.

Two lines of argument, however, challenge the validity of this conclusion. The first focuses on the possibility that the motivator employed in the study may not have been of sufficient strength to cause a change in writing behavior. This argument is refuted by the finding that when motivation was increased, the Comparison group showed large improvements on all tasks, while the CD group improved on all but the written output tests. It is illogical to argue that a motivator which is strong enough to cause such consistent and

equivalent change between groups should be of insufficient strength to change the written output of the CD group, unless there existed a motivation independent writing difficulty peculiar to that group.

The second argument is based on the possibility that neglect to counterbalance motivation across testing sessions may have resulted in a confounding of motivation and practice effects. Incentive was always introduced in the second session and, consequently, some of the observed score changes could have resulted from the practice afforded by the first testing session. Because of this confounding effect, it is impossible to determine to what extent the score changes may be attributed to practice and to motivation. In order to differentiate the effects of incentive and practice, it would be necessary to isolate the practice component by testing a third group with two identical sessions under normal motivation conditions.

It should be noted that while some of the observed score improvements may have been due to practice effects, such effects should produce equivalent changes between groups on all tests. There is no reason to suspect that either group would derive more or less benefit from practice than the other. Since improvement was found on all tests except the CD group's written output, it must be concluded that the CD children suffer from some a writing disability which is independent of practice and/or motivational influences.

The results indicate that there does exist, as hypothesised by

Heilman and Valenstein (1979), a group of children who have no verbal/linguistic deficits, but who are unable to organize and produce coherent written material. Although previous research appears to have identified the disorder which may now be labelled Coding Disability, it was encountered in the course of investigations of other syndromes and has never been addressed directly. Much of the relevant research has been done with adults suffering from various neurological problems. However, the findings of Heilman et al. (1973, 1974), Gordinier (1899), Assal et al. (1970), and Sugushita et al (1980) are generally comparable to those of the current study. All identified forms of dysgraphia existing in the absence of speech or reading disorders. The major significance of the similarity between the adult studies and the present investigation is in specifying the etiology of the Coding Disability. It was concluded from the adult studies that pure and apraxic dysgraphias were mediated by frontal lobe lesions. Extrapolating from this conclusion, it may be speculated that the Coding Disability results from similar defects, thus providing a convenient basis for further research into the origins of academic writing difficulties.

Only Levine et al. (1981), in their study of general writing disorders, described, in an academic setting, a form of apraxically mediated dysgraphia which may be the same as the Coding Disability. They identified two groups of children for whom the major impediment to writing was a type of dyslexia, and a third group whose dysgraphia appeared to be a function of a writing specific motor difficulty. It

is this third group which appears to be analogous to the Coding Disabled children of the present study. Levine et al. described behaviours such as poor pencil control and poor fine motor skills which may be characteristic of and linked to Coding Disability, but neglected to indicate to what extent the children who displayed these deficits showed any evidence of verbal/linguistic problems. If the apraxic dysgraphia identified by Levine and associates was independent of verbal difficulties, it can be argued that the observed disorder was the Coding Disability.

Working within a medical framework, Siegel and Feldman (1983) isolated a group of children similar to the Coding Disabled insofar as they exhibited academic underachievement, writing difficulties and low Coding scores without any form of dyslexia. However, Siegel and Feldman's subjects were also deficient in standardized arithmetic, short term memory and visual motor integration tests, as well as in standardized arithmetic and Digit Span subtests of the WISC-R. Problems were noted in both visual and oral output, but were more marked in written tests. The researchers concluded that there exists an interaction between disorders of writing, arithmetic, short term memory and visual motor integration since each of these four subproblems has a detrimental effect on the other three. While there exist striking similarities between the CD children and Siegel and Feldman's subjects, the major difference lies in the conception of the independence and causation of the Writing disorder experienced by both groups of children. In contrast to Siegel and Feldman's interactive

hypothesis, the Coding Disability is thought to result from a graphomotor specific apraxia and to be totally independent of disorders of memory or visuo-motor skills. Supporting this viewpoint is the finding that the CD children performed as well as the Comparison group on oral recall suggesting that they did not suffer any memory problems, and on the Block Design subtest suggesting that their visuo-spatial skills were on a par with those of the control children. It is impossible to determine whether there exists a relationship between Coding Disability and arithmetic problems. However the significant degree of similarity between the Coding Disability and the learning disability identified by Siegel and Feldman suggests that they may be variants of the same disorder.

While the current study attempted only to demonstrate the existence of the Coding Disability, the observations of the experimenter are of considerable relevance to both an in depth description of the disorder and suggestions for further research. No a priori hypotheses were formed to predict what types of specific motor difficulties could occur, but the subjective observations of the experimenter suggest a pattern of maladaptive writing behaviours which may be characteristic of the CD syndrome. It was noted that many children in the CD group displayed a form of graphomotor apraxia as they were writing paragraph summaries or completing the Coding subtest. As Levine et al. (1981) demonstrated, many of them displayed improper and awkward pencil grip. Several used a full fist, rather than a finger thumb grasp. Others exhibited a distal imperfection of

grasp, holding the pencil near its centre rather than at the point. As a result, pencil manipulation was extremely awkward and clumsy. Many of the children in the CD group also displayed poor fine motor skills as they wrote. While the comparison children manipulated their pencils with their fingers in the normal fashion, many of the CD subjects appeared incapable of such fine co-ordination. They often maintained stiff wrists and fingers and/or used their whole arms, pivoting at the shoulder, to move the pencil along the page. Writing was, therefore, ill formed, clumsy, and slow. In comparison, none of this awkwardness in pencil grip or graphomotor movement was noticed in the control children. While it may be argued that these difficulties are learned, prior research by Levine et al. (1981) suggests that they are hardwired.

It was also noted that, while many CD subjects displayed extreme awkwardness in their writing, they showed no apraxic symptoms when required to manipulate the blocks in the WISC-R Block Design test. Although Block Design may be mediated by verbal and/or analytic skills, strengths in these areas should facilitate only the cognitive solving of that solution, not the fine motor abilities required to physically construct that solution. The Coding Disabled and Comparison groups' equivalent scores on this test did not appear to reflect unusually fast problem solving skills balanced by unusually slow or awkward block manipulation on the part of the CD group. On the contrary, the CD children's fine motor co-ordination was excellent; movements were as rapid and accurate as those of the

comparison group. This suggests that the CD group's fine motor skills deficit is limited specifically to the movements involved in writing.

Additional subjective observation and examination of the children's behaviour during writing revealed further between groups differences. Although all children completed approximately the same number of paragraphs on the comprehension test, the relative proportions of time spent reading and writing appeared to vary widely between groups. The Control children spent about the same amount of time reading as they did writing their recall. The CD subjects, on the other hand, read the selections more rapidly than the control children. They then proceeded to spend prolonged amounts of time writing narrative recollections which were of lesser quality than those of the comparison subjects. It might be argued that this deficit could be the result of the CD children reading the paragraphs so fast that they did not assimilate and integrate all the information contained in the selection. This argument is refuted by the fact that these children clearly understood and integrated as much information as the Control subjects did, as evidenced by their equally good oral recall scores. The CD children understood the concepts and could express them verbally, but were unable to do so graphically.

Three of the CD children made spontaneous attempts to facilitate their written performance by verbalizing what they intended to write while in the act of writing. One child even went so far as to say each word and spell it aloud as he wrote it down. These three

children appeared to be unable to direct their hand movements internally, and they seemed to have to give verbal commands to their hands to write. While this coping strategy resulted in generally coherent written production, it remained very slow and labourious. Consequently, though the quality of the answer given was fairly high, the quantity was very much deficient. This has important ramifications for schoolwork, where the teacher is often interested in quantity, as well as quality of written work.

Upon examination of the types of errors which lead to missed points in written recall, two major problem areas, poverty of content and incoherence, emerge. Both groups of children earned only partial credit because their written recall did not include some of the main ideas in each paragraph. However, the children in the comparison group neglected the same main ideas in both their written and oral recall, suggesting that the information was either forgotten or never integrated during the initial reading. In contrast, the CD children neglected to include ideas in the written product that they did cover in their oral recall. They had grasped and integrated the main points of the paragraphs, but had not included them in their written output.

The other type of error which was characteristic of the CD children was incoherence. Many sentences were left unfinished and/or made no sense whatsoever. Even in cases where meaning remained broadly intact, words were omitted, resulting in some difficulty in penetrating the underlying ideas in the sentences. Alternatively,

words stood on their own with no context to explain or clarify their meanings. The written productions generally appeared to have little logical or conceptual organization. Often, this disorganization was so extensive that meaning was obscured to the point that it was impossible to give any credit. It can be argued that, although the CD children were able to organize their thoughts for verbal recall coherently, they were unable to do so for written production. As speculated by Weigl and Fradis (1977), the transcoding process may have broken down at the point of converting graphemes into graphomotor programmes. A similar argument, advanced by Dubois et al. (1969), would state that the children had trouble choosing the graphemes to construct the words, and were deficient in the spatio-temporal organization ability needed to write them. Finally, Levine et al. (1981) would argue that the CD subjects were so engrossed in concentrating on the arduous task of forming the letters and words that they were unable to maintain the unity or coherence of what they were trying to express. This argument could be used to account for problems of poverty of content as well. It is possible that the children who exhibited poverty of content were so intent on attempting to write the words of one thought correctly that, for the moment, they forgot other details of the paragraph.

Bad handwriting also appeared to be exclusive to the CD group. Although most of their writing was legible, the letters were very often large and ill formed, and looked more like the productions of Kindergarten students rather than fifth or sixth graders. This is

consistent with Heilman and Valenstein's (1979) postulated apraxic dysgraphia and lack of fine motor skills.

Spelling and grammatical errors were prevalent among both groups of children. There did not appear to be any particular pattern or type of spelling or grammar error which was characteristic of either group.

It must be reiterated that the foregoing description of error types and writing difficulties was based only on the experimenter's subjective observations during testing and on her ad hoc examination of the subjects' written output. They remain to be empirically validated. A more reliable scoring system must be developed to analyse written and oral recall, error types, and writing behaviours. Reliability could be improved by submitting for scoring the written recall profiles and tape recordings of the oral recall to one or more judges who are ignorant of the subjects' group membership. In addition to the scoring criteria established for oral and written recall in this study, guidelines should be set up to describe and govern the scoring of error types and aberrant writing styles.

A substantial body of literature exists which is aimed at the development and use of remedial handwriting strategies for children suffering from writing disabilities. Many of the writers and researchers who have studied the problem agree that the first step in any remediation programme is a task analysis of the desired act of

writing and/or a functional analysis of the problem behaviour.

Towle and Ginsburg (1975) and Towle (1978) argued that initial task analysis allows the teacher to examine the elemental components of a desired behaviour and to identify the point of breakdown for each individual child. The teacher is then able to design a highly specific remedial strategy aimed at teaching one particular elementary component of the desired behaviour. Once that component has been mastered, the remainder of the sequence is re-examined and other deficient components are dealt with in the same manner. Functional analysis contributes insofar as it identifies the environmental context in which the deviant behaviour is most likely to occur. It enables the teacher to take steps to restructure the settings to maximize potential for completion of the desired behaviour.

Applied to the problem at hand, task analysis breaks writing down into such components as deciding what to write, positioning the pencil, forming the first letter of the first word, then the second, and so on. If Coding Disability exists as hypothesised by Heilman and Valenstein, the breakdown should occur at the neurocognitive level, when the child attempts to organize the motor movements prerequisite to actually forming the letters. The first externally observable behavioural evidence of this deficit, that is, the point of breakdown as defined by the task analysis, appears when the child attempts to write the first letter of the first word. Therefore, a remedial

strategy with the ultimate goal of producing flowing coherent written work would have to address itself to helping the child learn to better organize his fine motor movements.

Towle (1978) suggests several teaching strategies which might be effective for this purpose. Sets of dot-to-dot pictures which start out large and somewhat coarse, but which get successively smaller, finer and more intricate, provide the child with graduated practice in using and sharpening fine motor skills. Alternatively, s/he may be asked to draw pictures between two parallel lines without ever crossing or touching these lines. As the child's fine motor skills improve, the boundaries of the drawing space become closer together, forcing the child to become more precise. This is somewhat similar to the Montessori approach which teaches fine motor and pencil control by having the child colour shapes which gradually become smaller and more intricate (Guyer, 1975). Finally, tracing pictures and letters which get progressively smaller serves the same end and leads naturally into functional writing. Once the child can cope with tracing small letters, the dotted prompts can be faded out, thus building independence of writing type movements.

The cursive writing techniques of Durbrow (1968), Gillingham and Stillman (1936) and others provide similar intensive practice in the use of fine motor skills to form letters. Concentrating on rapid, fluid production of written letters, this strategy involves the child first practicing series of easy upward looping letters like *l*

and *l* until s/he reaches a pre-set criterion of speed and accuracy. At that point, s/he goes on to slightly harder up and over letters such as *u*, and then on to the difficult up, over, and back letters like *n*. While, as Hanson (1976) points out, this method has the advantage of producing rhythmic, fluid, automatic writing, it requires that the child already have a reasonable degree of proficiency in fine motor skills. It is, therefore, inappropriate for children who are severely apraxic. However, when used in combination with Towle's suggestions, which are appropriate to a very low level of fine motor skill, cursive writing is certainly an excellent remediation strategy for those suffering from apraxic agraphia. Because it emphasises and teaches the production of smooth, automatic writing, it allows the individual the opportunity to organize his/her thoughts while he is writing, rather than to concentrate only on the actual act of forming the letters. Cursive writing should reduce significantly the errors of incoherence and poverty of content discussed earlier.

A similar approach, based on a functional analysis of the problem behaviour, was advanced by Stott (1978) and Mullins et al. (1972). They suggested that poor handwriting be described and treated in terms of its elemental faults. The most common of these include irregularity of slope, variation in letter size, irregularity of shape, and inability to stay on the line. Stott argued for the

identification and treatment of specific faults in handwriting. Instead of trying to develop new writing behaviours in addition to the old deficits, he advised using those deficits as the starting point for remediation. The rationale was that, by correcting the undesirable writing behaviour, defects are being eliminated at the same time that proper writing skills are being taught. Once isolated, the faults are remediated through the use of cursive writing techniques and graduated practice. In the final analysis, both Durbrow's and Stott's methods are equivalent. Only the theoretical orientation differs.

Two unusual techniques for treating writing difficulties are the typewriting method of Campbell (1973, 1976) and the biofeedback approach of Carter and Russell (1980). The former is based on the premise that, while dysgraphic children do not have the fine motor skills to manipulate a pencil properly, an effective remediation strategy could capitalize on using intact gross motor skills to hit typewriter keys. Campbell argues that this approach would eliminate the problem of ill formed, illegible letters while developing the use of visual, auditory, kinaesthetic and tactile senses in academic work. In a study of children suffering from both dyslexia and dysgraphia, she taught one group to use electric typewriters for all written work and left an untreated control group to use their pencils. After several weeks of this treatment, it was found that the experimental group had made significant progress in reading vocabulary skills. It was argued that use of the typewriter, a simpler movement than

writing, allowed the children to concentrate more on their word learning than on trying to put the words into written form. If this is extrapolated to the theory that Coding Disability results from the children not being able to concentrate on their written output because they are too intent on organizing their writing movements, it follows that use of the typewriter might allow them more freedom to think and to produce coherent narratives. It should be noted that Campbell cites research by Ayres (1965) which indicated that typewriting may circumvent apraxic responses which interfere with learning. Even though the "hunt and peck" method of the novice typist is slow, it is apparently less subject to apraxia than is writing per se.

Like Campbell (1973), Siegel and Feldman (1983) recommended the use of typewriters to avoid the problem of the child having to simultaneously organize and write his thoughts. In addition, they suggested that the academic assessment of a child with a Coding Disability be based more on oral than written output. This could be accomplished through the use of tape recorders; instead of writing a composition, the child could be required to dictate it into a tape which the teacher could review and evaluate at his/her leisure.

Carter and Russell (1980) suggest that dysgraphia with apraxia, in the absence of linguistic disorders, may result from severe graphomotor specific anxiety which disrupts motor skills. They suggest the use of biofeedback relaxation training to eliminate this maladaptive tension. Carter and Russell applied this technique to

four low achieving learning disabled children and observed, in each subject, significant improvements in reading, spelling and arithmetic, as well as in self-control, attention span and completion of seat work. In the present study, several of the CD children's Coding and Written recall scores actually decreased when motivation, and presumably arousal, was increased. The introduction of a relaxation programme might have improved performance by reducing arousal to an optimal level.

It remains to turn to the children themselves for a remediation strategy specific to their particular problem. A self instruction type approach (Meichenbaum, 1977; Kosiewicz et al., 1982), similar to that used by several of the children who attempted to direct their writing verbally, would probably be effective with repeated practice. Though it would, initially, result in very slow writing, with intensive practice, it could become automatic and internalized as the children developed motor programmes for writing.

The current study has fulfilled its objective insofar as it has demonstrated the existence of the Coding Disability. However, in doing so, it has raised many more questions about the etiology of the disorder, the specific behavioural components involved, the nature of the errors it causes, and the remediation strategies which might be effective. Because of the school curriculum's heavy emphasis on written output, greater understanding, and early identification and remediation, of the Coding Disability is of utmost importance to the

children so affected. Further research is needed from professionals in the fields of medicine, education, and psychology so that Caring Disabled children may learn to overcome their handicap and to perform to their maximum potential.

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APPENDIX A

Scoring Criteria

The PPVT and the Similarities, Vocabulary, Block Design, and Coding subtests of the WISC-R were administered and scored in the standard manner. The raw scores on the Wechsler subtests were converted into scaled scores, and the PPVT raw scores were analysed as stanines.

Administration procedures of the Gates-McGinitie Reading Comprehension test were altered to suit the purposes of the current investigation. Consequently, standardization was broken, and it was impossible to compare the subjects' raw scores with the test norms or to convert these scores into standard scores. Analysis was, therefore, performed on the raw scores.

Oral and written recall were evaluated on the basis of the number of discrete chunks of information recalled from each paragraph. The sentences in the selections were broken down by content into a number of phrases, each of which contained one item of information. For example the sentence 'The quick brown fox jumped over the sleeping dog' would have been broken down into 'The quick/brown/fox/jumped/over/the sleeping/dog', containing 7 separate ideas. Scoring was based on the number of items remembered by the child, and each item was scored as 1 or 0. One point was credited for each chunk of information recalled correctly, while zero was allotted for each item forgotten or remembered incorrectly. Scoring was not dependent on the child's being able to quote the passage verbatim, but rather relied upon his remembering the main ideas expressed in the selection.

Paraphrasing was acceptable and the child was not penalized either for giving additional information which was not presented in the original selection or for changing the sequence of events. An acceptable alternative for the previously quoted example might be "There was this beagle dog asleep in the grass by a tree. Along came a speedy brown fox and jumped over him."

General scoring criteria for that example are as follows:

1) quick

1 point - any word or phrase suggestive of fast movement (nimble, agile, swift, speedy, fast)

0 points - any word suggestive of slow or normal pace (lazy, slow)

2) brown

1 point - any shade of brown or logical modification of brown by another colour (beige, tan, reddish brown)

0 points - any other colour but brown

3) fox

1 point - any name commonly accepted as representing a fox, including those found in fairy tales (renard, Mr. Renard, Brer Fox)

0 points - any other animal (lion, wolf, dog)

4) jumped

1 point - hopped, leaped, sprung, cleared

0 points - fell, stumbled, walked

5) over

1 point - any word or phrase indicating that the jump completely cleared the dog (across)

0 points - any word suggesting that the fox in some way came into physical contact with the dog or failed to go over him (on top of, beside)

6) sleeping

1 point - any word suggesting a non-active and reclining position (snoring, resting, flaked out, lying, asleep)

0 points - any word suggesting an active or upright position (sitting, standing, barking, walking)

7) dog

1 point - any breed of dog or commonly accepted synonym for a dog (beagle, foxhound, dachshund, mutt, hound, puppy, canine)

0 points - any other animal or unspecified idiosyncratic name for a dog (cat, cow, Spot, Rover)

To facilitate rapid scoring, the dissected paragraphs were organized as a checklist against which oral and written recall could be evaluated. Oral recall was scored simultaneously, while written recall was scored while the child was reading the next selection. Points were tallied across paragraphs to produce the total oral and written recall scores.

APPENDIX B

Tables of Data

— U —

Table 1
Coding Disabled Group Test Scores Under Normal Motivation

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|------|-------|------|------|-------|-------|------|
| | | Sim | Voc | BLD | Cod | PPVT | WRc | ORc | MC |
| OW | 1 | 10 | 9 | 10 | 4 | 4 | 10 | 15 | 2 |
| | 2 | 12 | 10 | 11 | 2 | 2 | 12 | 20 | 4 |
| | 3 | 9 | 11 | 13 | 2 | 7 | 14 | 41 | 10 |
| | 4 | 11 | 10 | 11 | 6 | 5 | 13 | 55 | 16 |
| | 5 | 9 | 10 | 10 | 5 | 4 | 35 | 45 | 10 |
| | 6 | 7 | 5 | 5 | 5 | 2 | 10 | 10 | 1 |
| WO | 7 | 10 | 6 | 12 | 4 | 7 | 8 | 13 | 8 |
| | 8 | 12 | 8 | 12 | 12 | 5 | 29 | 45 | 12 |
| | 9 | 10 | 12 | 14 | 7 | 7 | 23 | 28 | 6 |
| | 10 | 6 | 8 | 2 | 9 | 4 | 39 | 53 | 11 |
| | 11 | 10 | 9 | 10 | 3 | 5 | 23 | 41 | 9 |
| | 12 | 12 | 10 | 13 | 3 | 5 | 15 | 34 | 5 |
| MEAN | | 9.00 | 9.00 | 10.25 | 5.17 | 4.75 | 19.67 | 34.83 | 8.33 |
| SD | | 2.83 | 2.00 | 3.47 | 2.98 | 1.71 | 10.34 | 16.70 | 4.38 |

NOTE: Abbreviations are used as follows:

Sim - Similarities
 Voc - Vocabulary
 BLD - Block Design
 Cod - Coding
 PPVT - Peabody Picture Vocabulary Test
 WRc - Written Recall
 ORc - Oral Recall
 MC - Multiple Choice
 OW - Order of Recall: Oral - Written
 WO - Order of Recall: Written - Oral

Table 2
Comparison Group Test Scores Under Normal Motivation

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|------|-------|------|------|-------|-------|------|
| | | Sim | Voc | BLD | Cod | PPVT | WRC | ORC | MC |
| OW | 1 | 11 | 12 | 17 | 13 | 6 | 36 | 37 | 9 |
| | 2 | 12 | 7 | 13 | 11 | 3 | 14 | 20 | 7 |
| | 3 | 10 | 8 | 11 | 6 | 2 | 21 | 26 | 8 |
| | 4 | 11 | 6 | 13 | 9 | 2 | 38 | 53 | 12 |
| | 5 | 5 | 6 | 7 | 8 | 1 | 7 | 14 | 5 |
| | 6 | 10 | 10 | 8 | 8 | 5 | 41 | 55 | 8 |
| WO | 7 | 9 | 5 | 13 | 11 | 4 | 10 | 20 | 6 |
| | 8 | 10 | 9 | 13 | 10 | 4 | 35 | 53 | 12 |
| | 9 | 9 | 8 | 12 | 6 | 4 | 7 | 10 | 1 |
| | 10 | 10 | 8 | 15 | 10 | 7 | 27 | 32 | 9 |
| | 11 | 9 | 10 | 8 | 10 | 5 | 34 | 26 | 11 |
| | 12 | 4 | 7 | 8 | 6 | 2 | 21 | 39 | 6 |
| MEAN | | 9.17 | 8.00 | 11.50 | 9.00 | 3.75 | 24.25 | 32.08 | 7.83 |
| SD | | 2.37 | 2.00 | 3.15 | 2.26 | 1.82 | 12.61 | 15.53 | 3.16 |

Table 3
Coding Disabled Group Test Scores Under High Motivation³

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|-------|-------|------|------|-------|-------|------|
| | | Sim | Voc | BID | Cod | PPVT | WRc | ORc | MC |
| OW | 1 | 11 | 8 | 12 | 3 | 5 | 11 | 19 | 4 |
| | 2 | 14 | 10 | 11 | 4 | 4 | 22 | 24 | 7 |
| | 3 | 10 | 11 | 15 | 4 | 8 | 12 | 49 | 10 |
| | 4 | 13 | 11 | 13 | 8 | 6 | 17 | 92 | 18 |
| | 5 | 11 | 11 | 12 | 5 | 4 | 41 | 57 | 10 |
| | 6 | 7 | 7 | 7 | 5 | 2 | 15 | 29 | 3 |
| WO | 7 | 14 | 14 | 11 | 2 | 8 | 13 | 26 | 9 |
| | 8 | 15 | 9 | 15 | 14 | 5 | 34 | 51 | 11 |
| | 9 | 14 | 15 | 19 | 10 | 7 | 31 | 39 | 7 |
| | 10 | 10 | 10 | 8 | 6 | 4 | 44 | 70 | 16 |
| | 11 | 14 | 10 | 14 | 5 | 5 | 32 | 64 | 11 |
| | 12 | 14 | 11 | 17 | 3 | 6 | 20 | 52 | 11 |
| MEAN | | 12.25 | 10.58 | 12.85 | 5.75 | 5.33 | 24.33 | 47.67 | 9.75 |
| SD | | 2.42 | 2.23 | 3.46 | 3.42 | 1.78 | 11.62 | 21.58 | 4.33 |

Table 4
Comparison Group Test Scores Under High Motivation

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|------|-------|-------|------|-------|-------|------|
| | | Sim | Voc | BID | Cod | PPVT | WRc | ORc | MC |
| OW | 1 | 16 | 14 | 19 | 15 | 7 | 56 | 60 | 9 |
| | 2 | 12 | 7 | 15 | 13 | 3 | 17 | 40 | 8 |
| | 3 | 9 | 8 | 15 | 9 | 3 | 33 | 39 | 10 |
| | 4 | 11 | 9 | 16 | 14 | 3 | 52 | 70 | 15 |
| | 5 | 6 | 6 | 9 | 11 | 2 | 8 | 20 | 6 |
| | 6 | 13 | 11 | 10 | 14 | 5 | 60 | 77 | 12 |
| WO | 7 | 10 | 3 | 17 | 15 | 5 | 20 | 28 | 8 |
| | 8 | 14 | 11 | 17 | 12 | 4 | 47 | 81 | 15 |
| | 9 | 12 | 9 | 18 | 10 | 4 | 13 | 23 | 3 |
| | 10 | 15 | 9 | 12 | 12 | 8 | 28 | 38 | 8 |
| | 11 | 13 | 10 | 14 | 11 | 5 | 48 | 48 | 10 |
| | 12 | 15 | 7 | 13 | 9 | 3 | 35 | 47 | 12 |
| MEAN | | 11.33 | 8.67 | 14.58 | 12.08 | 4.33 | 34.75 | 47.58 | 9.67 |
| SD | | 3.37 | 2.81 | 3.12 | 2.15 | 1.78 | 17.79 | 20.47 | 3.50 |

Table 5
Coding Disabled Group Change Scores

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|------|------|------|------|------|-------|------|
| | | Sim | Voc | BlD | Cod | PPVT | WRc | ORc | MC |
| OW | 1 | 1 | -1 | 2 | -1 | 1 | 1 | 4 | 2 |
| | 2 | 2 | 0 | 0 | 2 | 2 | 10 | 4 | 3 |
| | 3 | 1 | 0 | 2 | 2 | 1 | -2 | 8 | 0 |
| | 4 | 2 | 1 | 2 | 2 | 1 | 4 | 37 | 2 |
| | 5 | 2 | 1 | 2 | 0 | 0 | 6 | 12 | 0 |
| | 6 | 0 | 2 | 2 | 0 | 0 | 5 | 19 | 2 |
| WO | 7 | 4 | 8 | -1 | -2 | 1 | 5 | 13 | 1 |
| | 8 | 3 | 1 | 3 | 2 | 0 | 5 | 6 | -1 |
| | 9 | 4 | 3 | 5 | 3 | 0 | 8 | 11 | 1 |
| | 10 | 4 | 2 | 6 | -3 | 0 | 5 | 17 | 5 |
| | 11 | 4 | 1 | 4 | 2 | 0 | 9 | 23 | 2 |
| | 12 | 2 | 1 | 4 | 0 | 1 | 5 | 18 | 6 |
| MEAN | | 2.42 | 1.58 | 2.58 | 0.58 | 0.58 | 5.08 | 14.33 | 1.92 |
| SD | | 1.38 | 2.28 | 1.98 | 1.88 | 0.67 | 3.26 | 9.40 | 2.02 |

Table 6
Comparison Group Change Scores

| ORDER | SUB | Tests | | | | | | | |
|-------|-----|-------|------|------|------|------|-------|-------|------|
| | | Sim | Voc | BLD | Cod | PPVT | WRc | ORc | MC |
| OW | 1 | 5 | 2 | 2 | 2 | 1 | 20 | 23 | 0 |
| | 2 | 0 | 0 | 2 | 2 | 0 | 3 | 20 | 1 |
| | 3 | -1 | 0 | 4 | 3 | 1 | 12 | 13 | 2 |
| | 4 | 0 | 3 | 3 | 5 | 1 | 14 | 17 | 3 |
| | 5 | 1 | 0 | 2 | 3 | 1 | 1 | 6 | 1 |
| | 6 | 3 | 1 | 2 | 6 | 0 | 19 | 22 | 4 |
| WO | 7 | 1 | -2 | 4 | 4 | 1 | 10 | 8 | 2 |
| | 8 | 4 | 2 | 4 | 2 | 0 | 12 | 28 | 3 |
| | 9 | 3 | 1 | 6 | 4 | 0 | 6 | 13 | 2 |
| | 10 | 5 | 1 | -3 | 2 | -1 | 1 | 6 | -1 |
| | 11 | 4 | 0 | 6 | 1 | 0 | 24 | 22 | -1 |
| | 12 | 1 | 0 | 5 | 3 | 1 | 14 | 8 | 6 |
| MEAN | | 2.17 | 0.67 | 3.08 | 3.08 | 0.58 | 11.33 | 15.50 | 1.83 |
| SD | | 2.08 | 1.30 | 2.43 | 1.44 | 0.55 | 7.52 | 7.54 | 2.04 |

APPENDIX C

Summary of Statistical Analyses

Table 7

Wilks' Lambda and Univariate F-Ratio With 1 and 22 Degrees
of Freedom: Normal Motivation Condition

| Variable | Wilks' Lambda | F | Significance |
|----------|---------------|-------|--------------|
| Sim | 1.00 | 0.03 | 0.8 |
| Voc | 0.94 | 1.5 | 0.23 |
| BlD | 0.96 | 0.86 | 0.37 |
| Cod | 0.64 | 12.62 | 0.00 |
| PPVT | 0.92 | 1.93 | 0.18 |
| WEc | 0.96 | 0.95 | 0.34 |
| ORc | 0.99 | 0.17 | 0.68 |
| MC | 1.00 | 0.10 | 0.75 |

Table 8

Wilks' Lambda and Univariate F-Ratio With 1 and 22 Degrees
of Freedom: High Motivation Condition

| Variable | Wilks' Lambda | F | Significance |
|----------|---------------|-------|--------------|
| Sim | 0.97 | 0.58 | 0.45 |
| Voc | 0.87 | 3.43 | 0.08 |
| BLD | 0.93 | 1.69 | 0.21 |
| Cod | 0.43 | 29.55 | 0.00 |
| PPVT | 0.92 | 1.90 | 0.18 |
| WRc | 0.88 | 2.88 | 0.10 |
| ORc | 1.00 | 0.00 | 0.99 |
| MC | 1.00 | 0.1 | 0.96 |

Table 9

Wilks' Lambda and Univariate F-Ratio With 1 and 22 Degrees Of Freedom: Change Scores

| Variable | Wilks' Lambda | F | Significance |
|----------|---------------|-------|--------------|
| Sim | 1.00 | .12 | 0.73 |
| Voc | 0.947 | 1.47 | 0.24 |
| BLD | 0.99 | 0.33 | 0.59 |
| Cod | 0.62 | 13.34 | 0.00 |
| PPVT | 1.00 | 0.00 | 1.00 |
| WEc | 0.76 | 6.97 | 0.01 |
| ORc | 0.99 | 0.11 | 0.74 |
| MC | 1.00 | 0.01 | 0.92 |

Table 10

Mode of Recall as a Function of Presentation Order

| Order | Mode of Recall | | Row Total |
|----------------|----------------|---------|-----------|
| | Oral | Written | |
| Oral - Written | 185 | 93 | 278 |
| Written - Oral | 173 | 104 | 277 |
| Column Total | 358 | 197 | 555 |

Table 11

Chi-Squared Analysis of Presentation Order Effects on
Written and Oral Recall

| Chi squared | Degrees of Freedom | Significance |
|-------------|--------------------|--------------|
| 1.014 | 1 | 0.30 |

RNO>

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